manitoba neuroscience network

Genomic modifiers of the neurodegenerative disorder, Huntington disease

SEMINAR & VISITING SPEAKER SERIES

DATE
Friday, March 26, 2021
9:00AM

ZOOM LINK
https://us02web.zoom.us/j/83948652686?pwd=OFlURDh4dUZtbHh6K3JwaWdjTUcxQT09

MEETING ID
839 4865 2686

PASSCODE
547062

SPEAKER
Galen Wright, Ph.D.

Assistant Professor
Canada Research Chair in Neurogenomics
Department of Pharmacology & Therapeutics
Department of Biochemistry & Medical Genetics
Neuroscience Research Program
Kleysen Institute for Advanced Medicine
Health Sciences Centre and University of Manitoba

BIO

Dr. Wright started as an Assistant Professor at the University of Manitoba in April 2020 and is currently a Canada Research Chair in Neurogenomics. He completed his PhD in Genetics at Stellenbosch University, and received additional training in computational biology at the South African National Bioinformatics Institute. Dr. Wright then conducted a postdoctoral fellowship at the University of British Columbia. This fellowship led to the identification of highly predictive genetic biomarkers for various adverse drug reactions, including drug-induced neurotoxicities. Further, his precision medicine research has also involved the identification of clinically-relevant genetic variants which modify the age of onset of the neurodegenerative disorder, Huntington disease (HD).

RESEARCH

Huntington disease (HD) is a rare neurodegenerative disorder that is caused by a trinucleotide repeat expansion in the HTT gene. We have known for over 25 years that the length of this repeat is inversely correlated with disease onset. However, these models only explain approximately 70% of the variability in onset. The search for additional genomic modifiers of HD age of onset is therefore an active area of research, which has recently been facilitated by the assembly of large cohorts of affected individuals. These high throughput human genomic studies have consistently highlighted the importance of somatic repeat instability and genetic variation in DNA repair genes in modifying age of onset in the disorder. By providing new candidate therapeutic targets, these findings have significant implications both for HD, as well as for over 40 repeat mediated disorders.

OBJECTIVES

1) Understand HD genetics and the limitations of current age-of-onset prediction models

2) Learn about HD genomic modifiers throughout the genome

3) Identify the clinical and therapeutic implications of DNA repair-related modifiers

For more information:

T: 204-235-3939
E: info@manitobaneuroscience.ca

Health Sciences Centre Winnipeg
Hôpital St-Boniface Hospital RECHERCHE • RESEARCH
University of Manitoba